Psychodynamic therapy of obsessive-compulsive disorder: principles of a manual-guided approach

Obsessive-compulsive disorder (OCD) is a chronic disabling disorder characterized by recurrent obsessions and uncontrolled compulsions. Recent research suggests that OCD is more common than assumed before[1]. Cognitive-behavioral therapy and selective serotonin reuptake inhibitors have been shown to be equally efficacious in OCD[2], but with rates between 50% and 60% for response and 25% or below for remission[3]. Thus, further development of efficacious treatments is required.

Despite the long clinical tradition of describing and treating OCD from a psychodynamic perspective, no evidence-based psychodynamic treatment exists. Recent research on anxiety disorders, however, suggests that manual-guided short-term psychodynamic therapy (STPP) may be a promising approach[4]. Building on STPP for anxiety disorders, a model of STPP for OCD was developed which is based on Luborsky's supportive-expressive therapy[5]. The treatment consists of twelve modules which include both the characteristic elements of supportive-expressive therapy (i.e., focus on the core conflictual relationship theme, CCRT, and on the helping alliance) and additional disorder-specific treatment elements. In the following the treatment is briefly described.

At the beginning of treatment, the CCRT associated with the symptoms of OCD is assessed. A CCRT encompasses three components: a wish (W, e.g. aggressive or sexual impulses), a response from others (RO, e.g. to be condemned), and a response of the self (RS, e.g. obsessions and/or compulsions)[5]. Focusing on the CCRT, the therapist relates the patient's OCD symptoms (RS) to his or her wishes (or impulses and affects, W) and to the (expected) responses by others (RO). The CCRT is presented to the patient as his or her "OCD formula". This formula allows patients to understand their pattern of anxiety and OCD reactions. It translates the patient's symptoms into (internal and external) interpersonal relationships.

Enhancing the patient's cognitive and emotional understanding of his or her symptoms and of the underlying CCRT represents the expressive (interpretive) element of SE therapy[5]. An expressive intervention addressing the CCRT for Shakespeare's Lady Macbeth's compulsive washing may be[6]: "As we have seen your compulsive washing (RS) is related to your aggression, the murder of Duncan (W), and to your feelings of guilt (internalized RO). By your compulsive washing rituals, you are trying to make your deed undone and to get relief from your guilt feelings... By washing your hands again and again, you are replacing moral purity by physical cleanness".

During treatment, the CCRT and its components are worked through in present and past relationships, including the "here and now" relationship with the therapist. Consistent with available evidence[7], working through the CCRT can be expected to improve the patients' understanding of their conflicts, to reduce their OCD symptoms and to help them in developing more adaptive behaviors (RS). Both within and between sessions, patients are asked to work on their OCD formula, that is to monitor their emotions including their bodily components and to identify the components of the CCRT that lead to anxiety and OCD. Doing so, patients may achieve a better understanding and awareness of their OCD symptoms and a sense of control (i.e., not being helpless towards OCD), the latter being of particular importance for OCD patients.

Establishing a secure therapeutic alliance is regarded as the central ingredient of the supportive element of the intervention. Luborsky[5] has formulated several principles for establishing a secure alliance, e.g. conveying a sense of understanding and acceptance or recognizing the patient's growing ability to work on his or her problems in the same way the therapist does.

In order to tailor the treatment specifically to OCD, we integrated disorder-specific treatment elements that proved to be clinically helpful in OCD into the manual-guided model of STPP[8]. They encompass, for example:

- Differentiating between thinking and acting (e.g., "If you have sexual wishes towards these young women, this does not imply that you have actually committed adultery").
- Mitigating the rigid and hyper-strict super-ego (conscience) typically characteristic of OCD patients[8] (e.g., by not condemning the patient for his or her sexual or aggressive impulses; by encouraging the patient to resist against the super-ego's strict demands[7]). The super-ego can be regarded a part of the RO component of the CCRT.
- Freud's original recommendation to induce OCD patients to face the feared situation and to use the aroused experiences to work on the underlying conflict[9], in other words on the CCRT. The therapist may do so by saying, for example: "When you have these sexual (aggressive, etc.) thoughts towards young women, you get afraid that something terrible will happen to your wife. By carrying out your rituals you are trying to prevent this. We need to work on your expectation which entails not performing your rituals and tolerating the fear – and ultimately see what happens".

Further modules include: a) informing the patient about the disorder and the treatment, b) addressing ambivalence and setting treatment goals, c) establishing an encouraging inner dialogue, d) addressing (potential) non-response and resistance, and e) focusing on termination and relapse prevention.

We are planning to test the presented approach in a randomized controlled trial.

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1. Jacobi F, Hofler M, Siegert J et al. Int J Methods Psychiatr Res 2014;23: 304-19.
2. Romanelli RJ, Wu FM, Gamba R et al. Depress Anxiety 2014;31:641-52.
3. Foa EB, Liebowitz MR, Kozak MJ et al. Am J Psychiatry 2005;162:151-61.
4. Keefe JR, McCarthy KS, Dinger U et al. Clin Psychol Rev 2014;34:309-23.
5. Luborsky L. Principles of psychoanalytic psychotherapy. Manual for supportive-expressive treatment. New York: Basic Books, 1984.
6. Freud S. Obsessions and phobias. London: Hogarth Press, 1962/1895.
7. Crits-Christoph P, Luborsky L. In: Luborsky L, Crits-Christoph P (eds). Understanding transference: the CCRT method. New York: Basic Books, 1990:133-46.
8. Lang H. The inhibited rebel. Structure, psychodynamics and therapy of subjects with obsessive-compulsive disorders. Stuttgart: Klett-Cotta, 2015.
9. Freud S. Lines of advance in psycho-analytic therapy. London: Hogarth Press, 1955/1919.

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The social defeat hypothesis of schizophrenia: issues of measurement and reverse causality

Eleven years ago, two of us[1] published the social defeat hypothesis of schizophrenia, in an attempt to find a common denominator for several schizophrenia risk factors. The hypothesis posits that the long-term experience of being excluded from the majority group leads to an increased baseline activity and/or sensitization of the mesolimbic dopamine system, putting the individual at increased risk for the disorder[1,2].

The hypothesis may explain to a certain degree why a history of migration, membership of a disadvantaged ethnic minority group (e.g., African-American ethnicity), urban upbringing, low IQ, childhood trauma, drug abuse, hearing impairment, homosexuality[3,4], and perhaps also autism are schizophrenia risk factors.

We noted that the experience of defeat is neither a specific nor a sufficient or necessary risk factor for schizophrenia, and that other factors, including genetic vulnerability, co-participate in determining the nature of the outcome. Interestingly, neuroreceptor imaging studies reported evidence of dopamine sensitization in non-psychotic subjects with hearing impairment or with a history of childhood trauma, thus supporting the hypothesis[5,6].

However, there are at least two good reasons to criticize the hypothesis. First, it is difficult to measure social defeat in humans, because assessments based on interviews or questionnaires are biased by a tendency to give socially desirable replies. Second, one could argue that many children who go on to develop schizophrenia exhibit motor, cognitive and social impairments and that social defeat, therefore, is not a causal factor, but a consequence of a disorder in neurodevelopment, already present before the onset of psychosis and mainly driven by genetic factors.

As for the first issue, we recognize that the social defeat hypothesis is based on an interpretation of group comparisons (e.g., migrants versus natives, deaf subjects versus normal hearing individuals) and that we do not know with certainty whether individuals who develop schizophrenia are more "defeated" than others. This situation entails the risk of an ecological fallacy, which would be the case if, for example, successful migrants were found to be at equal risk of schizophrenia as non-successful migrants. However, we contend that the social defeat hypothesis is the most viable interpretation of the available data. The pattern of findings for ethnic minorities in Europe, for example, shows the highest risks for the least successful and most discriminated groups: African-Caribbeans and Black Africans in the UK, Inuit in Denmark and Moroccan-Dutch in the Netherlands.

As to the second point of criticism, we agree that schizophrenia likely "begins" long before the onset of psychosis. Studies of the Philadelphia Neurodevelopmental Cohort, for example, have shown that individuals aged 11 to 21 years who endorse psychotic symptoms (but do not meet the criteria for schizophrenia) are cognitively delayed, have a diminished whole brain grey matter volume, and grey matter volume deficits in frontal, temporal and parietal cortex[7]. It is true that these individuals are more likely to develop schizophrenia than others. However, given the fact that about 16% of all cohort members endorse psychotic symptoms, it is also evident that the majority will *not* develop the disorder and that motor, cognitive, social or anatomic impairments are merely risk factors or risk indicators of disorder, not hallmarks.

We propose that the epidemiology of schizophrenia supports a role for social exclusion, because it is unlikely that the genes that contribute to a defective neurodevelopment also code for migration, disadvantaged ethnic minority status, urban upbringing, low IQ, childhood trauma, drug abuse, homosexuality, hearing loss and autism. The social defeat hypothesis offers a more parsimonious explanation for this pattern of findings and deserves further development and testing.

First, since only two studies examined the risk of schizophrenia among individuals with a non-heterosexual orientation, further investigations of this topic are required. The hypothesis can also be tested in various other discriminated groups, such as those who are physically less attractive, who harbor a congenital or acquired handicap, a gender identity disorder, etc..

Second, it is important to examine whether "defeated" individuals who develop schizophrenia differ from other defeated subjects in the way they cope with defeat. Are they more likely to deny the very occurrence of defeat or do they attribute their problems to external causes? If they deny any problem, can implicit association tests reveal that they are implicitly aware of an inferior position?

Third, it is possible to conduct experiments in the laboratory. One can expose individuals to a negative evaluation or rejection and examine which subjects react by developing an increase in subclinical psychotic symptoms.